Exhibit 3

Expert Statement *(Kenneth R. Miller)*

Contents:

1) The Scientific Status of Evolutionary Theory
2) Biology by Miller & Levine
3) Language of the Dover Statement
4) "Intelligent Design" as a Scientific Alternative to Evolution
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Professional Background:

I earned a Bachelor of Science degree in Biology at Brown University (1970), and attended the University of Colorado on a National Defense Education Act Fellowship, earning my Ph. D. in Biology in 1974. I joined the faculty of Harvard University in 1974 as a Lecturer, and was promoted to Assistant Professor in 1976. In 1980, I accepted a position at Brown University, and was subsequently promoted to Associate Professor (1982) and Professor (1986). I am a cell biologist whose research centers on the structure and function of biological membranes and membrane proteins. I have published more than 50 research papers in scientific journals, including Nature, Scientific American, and Cell. I have also written a number of scientific reviews, commentary articles, and book reviews published in similar journals. Together with Joseph S. Levine, I am coauthor of a number of high school and college textbooks in general biology that are widely used throughout the United States. I am a member of the American Association for the Advancement of Science, the American Institute for Biological Sciences, and The American Society for Cell Biology, in which I chair the Education Committee.

to produce new and novel functions. According to the doctrine of irreducible complexity, however, this should not be possible. If the flagellum is indeed irreducibly complex, then removing just one part, let alone 10 or 15, should render what remains "by definition nonfunctional." Yet the TTSS is indeed fully-functional, even though it is missing most of the parts of the flagellum. The TTSS may be bad news for us, but for the bacteria that possess it, it is a truly valuable biochemical machine.

The existence of the TTSS in a wide variety of bacteria demonstrates that a small portion of the "irreducibly complex" flagellum can indeed carry out an important biological function. Since such a function is clearly favored by natural selection, the contention that the flagellum must be fully-assembled before any of its component parts can be useful is obviously incorrect. As a result, the principal biochemical argument for intelligent design, the contention that the bacterial flagellum is irreducibly complex, has failed.

As I noted in an article for Natural History magazine (Miller 2002), similar analyses can be described for each of the other systems proposed as examples of intelligent design. The evolution of the vertebrate blood clotting cascade, for example, has been described in detail by Hanumanthaiah et al (2002), Davidson et al (2003) and Jiang and Doolittle (2003). The evolution of antibody-based adaptive immmunity, one of the most complex systems in the body, has been elucidated as well. This work has taken place in many laboratories, and representative reports have appeared in papers by Lewis and Wu (2000), Market and Papavasiliou (2003), DuPasquier et al (2004), Zhou et al (2004), and Klein and Nikolaidis (2005). In addition, Nonanka and Yoshizaki (2004) were able to show how evolution produced the complement system, a complex and important part of the body's defenses against infection.

More generally, Long et al (2003) have reviewed the origin of new genes with novel functions, and have described 22 examples of such genes. Krem and DiCera (2002) have described the ways in which evolution produces that complex cascade-like pathways that function in signaling pathways associated with functions from blood clotting to signal transduction in development. Intelligent design bases its critique of evolution on the claim that new information cannot be produced by Darwinian mechanisms, and yet this claim has been repeatedly disproved by observations of novel pathways and enzymes that have arisen in the recent past. Prijambada et al (1995) described the ways in which Darwinian mechanisms produced nylonase, a new enzyme that breaks down the synthetic polymer nylon. Despite the claims of "design" advocates to the contrary, the ability of living organisms to response to environmental change by evolution is truly remarkable. Bacteria have even been able to evolve new pathways to break down 2,4-dinitrotolulene, the explosive compound in TNT (Johnson et al, 2002).

The Informational Challenge to Evolution

At first glance, William Dembski's case for intelligent design seems to follow a distinctly different strategy in dealing with biological complexity. His recent book, *No Free Lunch* (Dembski 2002a), lays out this case, using information theory and mathematics to show that life is the result of intelligent design. Dembski makes the assertion that living organisms contain what he calls "complex specified information" (CSI), and claims to have shown that the

evolutionary mechanism of natural selection cannot produce CSI. Therefore, any instance of CSI in a living organism must be the result of intelligent design. And living organisms, according to Dembski, are chock-full of CSI.

Dembski's arguments, couched in the language of information theory, are highly technical and are defended, almost exclusively, by reference to their utility in detecting information produced by human beings. These include phone and credit card numbers, symphonies, and artistic woodcuts, to name just a few. One might then expect that Dembski, having shown how the presence of CSI can be demonstrated in man made objects, would then turn to a variety of biological objects. Instead, he turns to just one such object, the bacterial flagellum.

Dembski offers his readers a calculation showing that the flagellum could not have possibly have evolved. Significantly, he begins that calculation by linking his arguments to those of Behe, writing: "I want therefore in this section to show how irreducible complexity is a special case of specified complexity, and in particular I want to sketch how one calculates the relevant probabilities needed to eliminate chance and infer design for such systems" (Dembski 2002a, p. 289). Dembski then tells us that an irreducibly complex system, like the flagellum, is a "discrete combinatorial object." What this means, as he explains, is that the probability of assembling such an object can be calculated by determining the probabilities that each of its components might have originated by chance, that they might have been localized to the same region of the cell, and that they would be assembled in precisely the right order. Dembski refers to these three probabilities as P_{orig}, P_{local}, and P_{config}, and he regards each of them as separate and independent (Dembski 2002a, p. 291).

This approach overlooks the fact that the last two probabilities are actually contained within the first. Localization and self-assembly of complex protein structures in prokaryotic cells are properties generally determined by signals built into the primary structures of the proteins themselves. The same is likely true for the amino acid sequences of the 30 or so protein components of the flagellum and the approximately 20 proteins involved in the flagellum's assembly (McNab 1999; Yonekura et al 2000). Therefore, if one gets the sequences of all the proteins right, localization and assembly will take care of themselves.

According to Dembski, evolution could still not construct the 30 proteins needed for the flagellum. His reason is that the probability of their assembly falls below what he terms the "universal probability bound." According to Dembski, the probability bound is a sensible allowance for the fact that highly improbable events do occur from time to time in nature. To allow for such events, he agrees that given enough time, any event with a probability larger than 10^{-150} might well take place. Therefore, if a sequence of events, such as a presumed evolutionary pathway, has a calculated probability less than 10^{-150}, we may conclude that the pathway is impossible. If the calculated probability is greater than 10^{-150}, it's possible (even if unlikely).

When Dembski turns his attention to the chances of evolving the 30 proteins of the bacterial flagellum, he makes what he regards as a generous assumption. Guessing that each of the proteins of the flagellum have about 300 amino acids, one might calculate that the chances of getting just one such protein to assemble from "random" evolutionary processes would be 20^{-300},

since there are 20 amino acids specified by the genetic code. Dembski, however, concedes that proteins need not get the *exact* amino acid sequence right in order to be functional, so he cuts the odds to just 20^{-30}, which he tells his readers is "on the order of 10^{-39}" (Dembski 2002a, p. 301). Since the flagellum requires 30 such proteins, he explains that 30 such probabilities "will all need to be multiplied to form the origination probability"(Dembski 2002a, p. 301). That would give us an origination probability for the flagellum of 10^{-1170}, far below the universal probability bound. This is presented as proof that flagellum couldn't have evolved, and therefore must be the product of design.

In contrast to this confident conclusion, a careful analysis of the way in which Dembski calculates the probability of an evolutionary origin for the flagellum shows how little biology actually stands behind those numbers. His computation calculates only the probability of spontaneous, random assembly for each of the proteins of the flagellum. Having come up with a probability value on the order of 10^{-1170}, he assures us that he has shown the flagellum to be unevolvable. This conclusion, of course, fits comfortably with his view that "The Darwinian mechanism is powerless to produce irreducibly complex systems..." (Dembski 2002a, p. 289).

However complex Dembski's analysis, the scientific problem with his calculations is almost too easy to spot. By treating the flagellum as a "discrete combinatorial object" he has shown only that it is unlikely that the parts of flagellum could assemble spontaneously. Unfortunately for his argument, no scientist has ever proposed that the flagellum or any other complex object evolved that way. Dembski, therefore, has constructed a classic "straw man" and addressed it away with an irrelevant calculation.

By treating the flagellum as a discrete combinatorial object he has assumed in his calculation that no subset of the 30 or so proteins of the flagellum could have biological activity. As we have already seen, this is wrong. Nearly a third of those proteins are closely related to components of the TTSS, which does indeed have biological activity. A calculation that ignores that fact has no scientific validity.

More importantly, Dembski's willingness to ignore the TTSS lays bare the underlying assumption of his entire approach towards the calculation of probabilities and the detection of "design." *He assumes what he is trying to prove.*

According to Dembski, the detection of "design" requires that an object display complexity that could not be produced by what he calls "natural causes." In order to do that, one must first examine all of the possibilities by which an object, like the flagellum, might have been generated naturally. Dembski and Behe, of course, come to the conclusion that there are no such natural causes. But how did they determine that? In fact, this "conclusion" is an unsupported assumption upon which all of his calculations depend. Suppose that there are such causes, but one simply happened not to think of them? Dembski actually seems to realize that this is a serious problem. He writes: "Now it can happen that we may not know enough to determine all the relevant chance hypotheses. Alternatively, we might think we know the relevant chance hypotheses, but later discover that we missed a crucial one. In the one case a design inference could not even get going; in the other, it would be mistaken" (Dembski 2002a, p. 123 (note 80)).

What Dembski is telling us is that in order to "detect" design in a biological object one must first come to the conclusion that the object *could not* have been produced by any "relevant chance hypotheses" (meaning evolution). Then, and only then, are Dembski's calculations brought into play. Stated more bluntly, what this really means is that the "method" first involves *assuming the absence* of an evolutionary pathway leading to the object, followed by a calculation "proving" the impossibility of spontaneous assembly. This faulty *a priori* reasoning is exactly the sort of logic upon which the new "science" of intelligent design has been constructed.

Not surprisingly, scientific reviewers have not missed this point — Dembski's arguments have been repeatedly criticized on this issue and on many others (Orr 2002; Charlesworth 2002; Padian 2002).

The Origin of Biological Information

Arguments in favor of "design" are often predicated on the statement that living organisms contain large quantities of biological information (which is true) and that no natural process can account for the presence of this information (which is false). They then conclude that the existence of such information is evidence for design.

Such arguments ignore a wealth of research and scholarship on the origins of biological information. In reality, evolutionary mechanisms that can generate increased complexity and biological information are very well understood, and are described in many research papers. Adami *et al* (2000) described a carefully-controlled model system in which increases in information are driven by repeated rounds of reproduction, mutation, and selection, the same forces that drive evolutionary change in nature. Adami's system mimics the evolutionary process in remarkable detail, as highlighted in a 2003 article in Nature (Lenski *et al* 2003). Thomas Schneider of the National Institutes of Health has come to similar conclusions with respect to information based in nucleic acids (Schneider 2000).

Specific experiments on a variety of living organisms have shown that information does indeed arise through distinctly Darwinian mechanisms. The supporting evidence includes a number of studies on gene duplication (Brown *et al*, 2003; Ohta, 2003; Lynch & Conery, 2000; Hughes & Freeman, 2003), as well as experiments in which organisms have responded to adverse environmental conditions by increasing the information content of their DNA (Lenski, 1995; Papadopoulos *et al*, 1999; Riehle *et al*, 2001).

The origin of biological information, as nearly all of these scientists have pointed out, is explained by the mechanism of evolution itself. Variation in the information content of living organisms arises by means of mutations, a few of which increase information content. Natural selection then chooses those variations best-suited to the environment, "fixing" the increased information in the genome. The energetic price that such increases in information entail is considerable, but is fully accounted by the great cost of unsuccessful variants in the struggle for existence. To pretend otherwise, as the intelligent design movement has, is unfortunate and misleading.